Tuesday, March 5, 1991
2:00PM–3:30PM, Room 254, West Concourse
Clinical Conditions Affecting Cardiac Performance

2:00

TRANSESOPHAGEAL DOPPLER ECHOCARDIOGRAPHY DURING CARDIOPULMONARY RESUSCITATION IN HUMANS

TR Porter, J Ornato, E Racht, D Williams, E Jones, C Guard, J V Nixon, Med Coll of Virginia, Richmond, VA

The assessment of left (LV) and right (RV) ventricular function and mitral valve position during chest compression while administering closed chest CPR in humans provides details of the adequacy of chest compressions and whether cardiac or thoracic pump mechanisms are responsible for forward flow. We assessed LV and RV fractional shortening (FS) and mitral valve function with transesophageal color Doppler Echocardiography (TEE) in ten patients during CPR (5 asystole, 3 pulseless ventricular tachycardia or ventricular fibrillation, and 2 with electromechanical dissociation). Chest compression and ventilation were performed by a computer controlled mechanical Thumper. During standard CPR at a rate of 90/minute, RVFS exceeded LVFS (55±3% RV versus 26±3% LV, p<0.01). At a constant compression heart rate of 90, increasing piston force of compression from 90 to 140 pounds per square inch (n=5) increased LVFS significantly (23±5% to 30±4%, p<0.05), while RVFS did not change (57 ±4% to 53±4%, p=NS). When the underlying rhythm was asystole or an organized rhythm with electromechanical dissociation, mitral valve position was closed during piston compression. However, during pulseless ventricular tachycardia or ventricular fibrillation, mitral valve position was independent of chest compression or there was color Doppler evidence of mitral regurgitation. TEE during CPR demonstrates a) that RV fractional shortening exceeds LV fractional shortening; b) with increasing force of piston compression there is a significant improvement in left ventricular fractional shortening; and c) mitral valve position during chest compression appears to be a function of the underlying cardiac rhythm.

2:15

LONG TERM IMPROVEMENT IN LEFT VENTRICULAR FUNCTION FOLLOWING CORONARY THROMBOLYSIS.

A. Teddy Weiss, Paula Weiss, Mervyn S. Gotsman, Hadassah Hospitals, Jerusalem, Israel.

Thrombolytic therapy for acute myocardial infarction improves immediate mortality but its long term impact on left ventricular function is unknown. In order to evaluate changes in left ventricular function we performed, prospectively, serial nuclear ventriculography on day 1(a), 1 month (b), and 3 years (c) following AMI in 50 patients who received thrombolytic therapy within 4 hours of chest pain onset. Reperfusion was assessed clinically and at angiography on day 6. Group I (34 pts) had successful reperfusion while Group II (16 pts) failed to open or reinfarcted. Global LV function was calculated (EF%) and regional wall motion (WM) was visually scored in the infarct related area.

Results:

	EF(a)	EF(b)	EF(c)	RWM(a)	RWM(b)	RWM(c)
I:	56±12	59±11	61±11*	11±4	12±4	14±4**
II:	53±23	45±19	38±16**	9±6	8±5	6±4**

*P<.05 for (a) vs (c)
**P<.001 for (a) vs (c)

Serial nuclear ventriculography shows that in successful thrombolysis there is an early and long term improvement in regional and global LV function, versus a significant deterioration over time in failed reperfusion or reinfarction.

2:30

IMPROVEMENT IN RIGHT AND LEFT VENTRICULAR DIASTOLIC FUNCTION WITH REGRESSION OF HYPERTENSIVE CARDIAC HYPERTROPHY

Gabriel Habib, William A. Zoghbi, James K. Alexander, Miguel A. Quinones, Robert Roberts, Baylor College of Medicine, Houston, Tx

Regression of hypertensive cardiac hypertrophy has been documented echocardiographically with some anti-hypertensive drugs. However, it remains to be determined whether regression of cardiac hypertrophy is associated with improvement in ventricular diastolic filling dynamics in treated hypertensive patients. Accordingly, serial M-mode echocardiography and Doppler mitral and tricuspid inflow velocities were obtained in 17 hypertensive male subjects (age 32-59 years; diastolic blood pressure 95-115 mmHg) prior to, 1 month, 2 and 3 months following therapy with 30-150 mg/day of nifedipine XL. All patients achieved diastolic blood pressure <90 mmHg or a reduction of ≥10 mmHg compared to baseline. Left ventricular mass index (LVMI by M-mode), early (E) and late (A) Doppler inflow velocities were determined without knowledge of patients or sequence of studies. Study results are as follows:

	Baseline	1 Month	2 Months	3 Months
Diastolic BP, mmHg	109±6	94±7*	95±5*	94±6*
HR, bpm	74±9	78±8	75±9	76±8
LVMI, g/m2	161±36	131±30*	126±30*	110±20*
Mitral E/A	1.00±0.17	1.16±0.25*	1.34±0.33*	1.33±0.25*
Tricuspid E/A	1.07±0.29	1.31±0.36*	1.58±0.33*	1.78±0.34*

*p<0.05 vs baseline

Changes in mitral and tricuspid E/A ratios reflected predominantly changes in peak E velocity. Similar directional changes were observed for first one-third and first one-half filling fractions of both ventricles.

Conclusions: Following normalization of blood pressure, gradual reduction in cardiac hypertrophy was documented and was accompanied by progressive improvement in left and right ventricular filling dynamics. The improvement in right ventricular filling dynamics supports the concept that these changes are not due to altered loading conditions but due to improved myocardial relaxation.

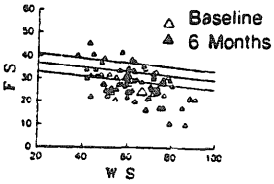
2:45

ABNORMAL LEFT VENTRICULAR FUNCTION IN PATIENTS WITH MITRAL STENOSIS IMPROVES FOLLOWING VALVULOPLASTY.

Prabodh M. Mehta, Joshua Wynne, Vincent P. Reyes, B. Soma Raju, D.N. Kumar, K. Srinath, Priscilla Peters, Zoltan G. Turi for the Nizam's Institute Wayne-State University Valvuloplasty Study Group, Hyderabad, India & Detroit, MI.

Decreased LV function in pts with mitral stenosis(MS) has been variously ascribed to either decreased preload or intrinsic contractile dysfunction. Also, the course of LV function following relief of mitral stenosis has not been well studied. We prospectively evaluated LV function in 60 pts with severe MS undergoing either percutaneous balloon or open surgical valvuloplasty(V). Cardiac catheterization carotid pulse and 2D/Doppler echo were performed at baseline(B) and 6 months (6m) after V. Patients with > mild mitral regurgitation(n=13), and suboptimal echo(n=8) were excluded. Mean±SEM

	B	6m
MVA	0.9±0.04	2.4±0.13*
EDVI	52.2±1.9	51.0±1.6
ESVI	26.2±1.1	20.5±0.9*
FS	24.8±0.9	31.8±1.0*
WS	66.1±2.0	60.0±2.0*
FSI	31.1±0.9	37.5±0.9*
Vcfc	0.7±0.03	0.9±0.03*
WS/ESVI	2.6±0.1	3.1±0.1*



*=p<0.008, EDVI=end diastolic volume index, ESVI=end systolic volume index, FS=fractional shortening, FSI=FS indexed for WS, MVA=mitral valve area, Vcfc=rate corrected mean velocity of circumferential fiber shortening, WS=end systolic wall stress. At baseline 30 of 39 pts(77%) were below the published normal range for FS vs. WS compared to 11 of 39 pts(28%) at 6m(p<0.001). Conclusion: (1) LV function is abnormal in many pts with severe MS; (2) LV function improves 6m following V and can not be explained by changes in loading conditions.